by computed tomography—on cardiovascular events after TAVR (2). An impaired survival was clearly evidenced in patients with a calcified aorta independently of usual confounders.

The study by Yotti et al. (1) also suggests that the usual tools would be unable to assess vascular impedance while the aorta is unloaded because of AS. It is rather challenging to separate valvular and vascular functions (3) and evidently, although very accurate, the approach used by Yotti et al. (1) could not be used routinely. Quantifying AAC on the systematic pre-TAVR computed tomography is an easy way to estimate aortic stiffness that should be more appropriate in the presence of AS because it is pressure independent.

We would like to emphasize the critical role of vascular load after TAVR while it is currently neglected. This deserves, to our view, implementing a simple stiffness index (e.g., AAC), in the pre-TAVR work-up. On top of being a stratifying variable that should be considered to refine the medical decision, high vascular load represents a potential avenue for new destiffening strategies and prompts us to target high blood pressure.

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REPLY: Aortic Stiffness

Complex Evaluation But Major Prognostic Significance Before TAVR

We very much appreciate the interest of Dr. Harbaoui and colleagues in our work (1). Their letter further elaborates on the clinical relevance that systemic vascular load may have in patients with aortic stenosis (AS) undergoing valve replacement. In this regard, we would like to emphasize that our study was designed to address specifically the impact of the stenotic valve on measurements of vascular load. For this purpose, we combined state-of-the-art measurement and signal-processing techniques with the mechanistic model of acute relief of the valvular load provided by transcatheter aortic valve replacement (TAVR). Albeit limited to a small sample size, we provided ancillary follow-up data suggesting a potential link between the acute vascular changes and the clinical functional improvement after the intervention (2). This should be interpreted only as a hypothesis-driven finding, which must be confirmed in a large clinical cohort. However, the highly sophisticated methods we used in our study are obviously nonsuitable for this purpose.

Systemic arterial compliance can be accurately approximated noninvasively using echocardiography and sphygmomanometry in patients without AS (3), and has proved to be a very powerful predictor of cardiovascular events in a number of populations. Data from our study (2) validates the noninvasive surrogate of systemic arterial compliance in patients with AS (intraclass correlation coefficient = 0.82 and 0.80 vs. the gold-standard invasive exponential decay and the diastolic area methods, respectively; n = 46 pooled pre- and post-TAVR data). Alternative indices based on tonometry (4) and ultrasound-based wave-intensity analyses (5) are also proven predictors of cardiovascular events, but have not been validated in the presence of AS. Quantifying the degree of ascending aortic calcification as proposed by Harbaoui et al. is an interesting surrogate of arterial stiffness because it is particularly well suited to fit in the screening workflow of patients considered for valve replacement, but more evidence supporting its clinical value is needed.

We believe the results of our findings suggest that metrics of vascular load should be incorporated to optimize patient periprocedural management. Long-term prospective studies will finally establish the prognostic value of measuring the ventricular-vascular interaction before and after valve interventions. Particularly important is to clarify the confusion caused by diastolic dysfunction, kidney disease, age, and coronary artery disease, amongst other highly prevalent conditions in patients with AS. Only when such information becomes available it shall be possible to establish how indices of vascular load may aid selecting the best management strategy for patients with AS.

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